



King's Research Portal

DOI:

[10.1016/j.ejvs.2018.08.042](https://doi.org/10.1016/j.ejvs.2018.08.042)

Document Version

Peer reviewed version

[Link to publication record in King's Research Portal](#)

Citation for published version (APA):

Clough, R. E., Barillà, D., Delsart, P., Ledieu, G., Spear, R., Crichton, S., Mounier Vehier, C., Peacock, J., Sobocinski, J., & Haulon, S. (2018). Long-term Survival and Risk Analysis in 136 Consecutive Patients With Type B Aortic Dissection Presenting to a Single Centre Over an 11 Year Period. *European Journal of Vascular and Endovascular Surgery*. <https://doi.org/10.1016/j.ejvs.2018.08.042>

Citing this paper

Please note that where the full-text provided on King's Research Portal is the Author Accepted Manuscript or Post-Print version this may differ from the final Published version. If citing, it is advised that you check and use the publisher's definitive version for pagination, volume/issue, and date of publication details. And where the final published version is provided on the Research Portal, if citing you are again advised to check the publisher's website for any subsequent corrections.

General rights

Copyright and moral rights for the publications made accessible in the Research Portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognize and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the Research Portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the Research Portal

Take down policy

If you believe that this document breaches copyright please contact librarypure@kcl.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.

1 **Long-term survival and risk analysis in 136 consecutive patients with type**
2 **B aortic dissection presenting to a single centre over an 11-year period**

3

4 Rachel E Clough MD PhD^{1,2}, David Barillà MD¹, Pascal Delsart MD¹, Guillaume
5 Ledieu MD¹, Rafaele Spear MD PhD¹, Siobhan Crichton PhD³, Claire Mounier
6 Vehier MD PhD¹, Janet L Peacock PhD³, Jonathan Sobocinski MD PhD¹, Stephan
7 Haulon MD PhD^{1,4}

8

9 ¹Aortic Centre, CHRU de Lille, France

10 ²Division of Imaging Sciences and Biomedical Engineering, King's College London

11 ³Division of Health and Social Care Research, King's College London,

12 ⁴ Aortic Centre, Hôpital Marie Lannelongue, Le Plessis-Robinson, Université Paris
13 Sud, France

14

15 Corresponding author

16 Stéphane HAULON

17 Aortic Centre, Department of Aortic and Vascular Surgery,

18 Hôpital Marie Lannelongue, Le Plessis-Robinson, INSERM UMR_S 999,

19 Université Paris Sud, France

20 Email : s.haulon@ccml.fr

21

22 **Article type:** Original article

23 **Running head:** Survival and risk analysis in aortic dissection

24

25 **What does this study add to the existing literature and how will it influence**
26 **future clinical practice?**

27 The management of patients with type B aortic dissection is complex. A number
28 of cases series have been published to try to better understand these patients but
29 they are limited by their small size. IRAD is a highly valuable resource but pools
30 data from a number of centres across the world and is therefore limited by the
31 heterogeneity of the data. This manuscript describes the findings of the largest
32 single centre series published to date and provides new insights into the
33 management of these complex patients.

34

35 **Abstract**

36 *Objectives:* To evaluate in patients with acute type B aortic dissection the results
37 of medical and endovascular treatment in large single centre experience and to
38 investigate the clinical and imaging features on presentation that relate to poor
39 outcome.

40

41 *Design:* Retrospective analysis of prospectively collected clinical and CT imaging
42 data.

43

44 *Materials:* 136 consecutive patients with acute type B aortic dissection were
45 included in the study over an 11-year period.

46

47 *Methods:* Characteristics of patients receiving endovascular (complicated) or
48 medical treatment (uncomplicated) were compared. Kaplan Meier estimators
49 were used to estimate cumulative overall survival and survival free of aortic
50 events. Factors associated with overall and aortic event free survival were also
51 explored using Cox-proportional hazards models.

52

53 *Results:* The mean follow-up was 51 months (1-132), during which time 33
54 deaths and 48 aortic events occurred. At one year and five years overall survival
55 was 94.0% and 74.8% respectively and freedom from aortic events was 75.6%
56 and 58.7%. There was no difference in all cause survival and aortic event-free
57 survival at one and five years between the patients treated endovascularly and
58 those receiving medical treatment alone. Risk analysis for aortic events
59 demonstrated the maximum size of the proximal entry tear, the maximum

60 thoracic aortic diameter and the thoracic aortic false lumen maximum diameter
61 to have a significant effect on the incidence of aortic events.

62

63 *Conclusions:* Active management of patients with type B aortic dissection results
64 in good long-term survival even in the presence of features traditionally
65 associated with adverse outcomes. All patients require close lifetime surveillance
66 as aortic events, even after endografting, continue to occur during follow-up.

67

68 *Keywords:* Survival, risk analysis, aortic dissection

69

70 **Introduction**

71 Type B aortic dissection is a complex clinical entity. In the presence of
72 complications such as rupture and end-organ ischaemia urgent endovascular
73 intervention is required.(1) Uncomplicated cases are currently treated medically,
74 with active management of blood pressure, to try to prevent complications
75 during follow-up such as extension of the dissection, aneurysm formation, and
76 rupture.

77

78 Previous series have however demonstrated that even in the presence of good
79 blood pressure control survival is poor, with only 50-70% of patients alive at 5
80 years, with a delayed expansion of the false lumen in 20-50% of patients at 4-
81 years.(2-4) Many of the deaths that occur during follow up are aortic related.

82

83 A number of groups, largely in Asian patient populations, have tried to identify
84 clinical and anatomical features that could be measured on presentation and be
85 used to predict outcome, and therefore identify patients at high risk of aortic
86 events and death during follow-up.(5) The majority of these studies have been
87 performed in small series (<40 patients), with results from some studies
88 contradicting the results of others and so currently there is no reliable method to
89 identify these high-risk patients.

90

91 There is growing interest in early endovascular treatment of patients with type B
92 aortic dissection to prevent aortic events during follow up and therefore improve
93 survival. It is hoped that treating patients early will produce the best outcomes

because the aorta has plasticity and is therefore likely to undergo positive aortic remodelling following stent graft insertion.(6)

We have an 11-year experience in the management of patients with type B aortic dissection using best medical treatment for uncomplicated and thoracic endovascular repair (TEVAR) for complicated dissection. The aim of this study was to evaluate the results that can be achieved with medical and endovascular treatment in these patients in a large single centre experience and to investigate the features on presentation that relate to a poor outcome.

Materials and methods

Study design

136 consecutive patients with acute type B aortic dissection were included in the study over an eleven-year period. All patients were managed in a high dependency setting with medical management implemented by medical specialists trained in the management of hypertension. Patients that presented with evidence of aortic rupture, end-organ ischaemia or on-going pain despite good blood pressure control were defined as complicated and treated endovascularly, those without these features were treated with best medical therapy. Patients were excluded if the aortic dissection was secondary to trauma or iatrogenic injury. Patients were categorised into groups based upon their treatment. All patients were followed up by both a cardiologist with a special interest in aortic dissection and hypertension and a vascular surgeon.

Endovascular repair

Thoracic endovascular repair was performed in a standard operating room with mobile C-arm or in a hybrid operating room using percutaneous vascular access whenever possible. Left common carotid to left subclavian artery (LSCA) bypass or transposition was considered if the stent graft covered the origin of the LSCA. Absolute indications for LSCA revascularisation were: left internal mammary artery bypass graft; diminutive, atretic or absent right vertebral artery; left arm arterio-venous fistula for haemodialysis; patent left axillo-femoral bypass graft; and dominant left vertebral artery. These procedures were performed prior to, or at the time of the TEVAR procedure, depending upon the level of urgency of the case. The stent graft was sized to the proximal un-dissected aorta with 10-

15% oversizing. In cases of rupture the stent grafts were positioned to cover the thoracic aorta from proximal to the primary entry tear to the level of the coeliac trunk.

Best medical therapy

Best medical therapy was implemented using a combination of antihypertensive medications. Our preferred stepwise approach was as follows: β -blocker, ACE-inhibitor, calcium channel blocker then diuretics with a final option of alpha-receptor blocker and/or a centrally acting antihypertensive agent. Blood pressure control was based on the European Society of Cardiology guidelines with a target blood pressure of below 135/80 mmHg.

Image analysis

Computed tomography (CT) angiographic images of the aorta were acquired on presentation and during follow-up using an iodinated contrast agent. All images were analysed on a dedicated Aquarius iNtuition workstation (TeraRecon, Foster City, Calif) by two vascular surgeons experienced in the endograft planning for aortic dissection. The arterial phase images, reconstructed to ≤ 1 mm, were evaluated and the number of entry tears, the size of the primary entry tear, the length of the dissection, the amount of thrombosis in the false lumen and the dimensions of the aorta and the true and false lumen were recorded. The true lumen was identified as the lumen continuous with the proximal undissected aorta. The maximum diameter of the primary entry tear was measured using multi-planar reformatted (MPR) images, false lumen thrombosis was assumed to be present when there was absence of contrast enhancement and volumes were

calculated by segmentation and summation over contiguous slices. The following were recorded: i. the maximum aortic diameter; ii. the true and false lumen diameters in the thoracic aorta at the level of the inferior pulmonary vein; and iii. the true and false lumen diameters in the abdominal section at the level of the IMA (or at the mid-point of the third lumbar vertebra if the IMA could not be seen). All diameter measurements were made using MPR images to ensure precision.

Statistical analysis

Characteristics of patients receiving endovascular or medical treatment alone were summarised within each group and compared using Mann-Whitney, chi-squared or fisher's exact test, as appropriate. Kaplan Meier estimators were used to estimate cumulative overall survival and survival free of aortic events with 95% confidence intervals at one and 5 years after admission. Aortic events were rupture, extension of the dissection and further intervention. Kaplan Meier estimators were also calculated within subgroups of patients. Factors associated with overall and aortic event free survival were also explored using Cox-proportional hazards models. Due to small sample sizes, for categorical data the estimation of one and five year survival rates and the application of Cox models were restricted to variables with at least 20% of patients in each subgroup. Where Cox models indicated a statistically significant association Kaplan Meier curves were plotted. For continuous variables cut points were selected that divided the data into three approximately equal sized groups and Kaplan Meier curves estimates for each of these groups. Analysis was conducted using STATA 13MP and p-values <0.05 considered statistically significant.

Results

Baseline characteristics

The baseline characteristics for the 136 patients are summarised in Table 1. The average age at admission was 61.7 years and the majority of the cohort were male (77.2%). Seventy-one per cent of the cohort had hypertension, 9.6% had diabetes and 5.9% had known connective tissue disease. Sixty-four patients presented with complications and were treated endovascularly. The most common complication was end-organ ischaemia (n=45); aortic rupture was present in 17 cases and 5 patients were treated for on-going pain despite good blood pressure control. Three patients had both rupture and end-organ ischaemia. The patients that were treated endovascularly were significantly younger than the patients treated with medical treatment alone (mean age 58.5 versus 64.5 years). β -blocker was the most commonly used antihypertensive agent to treat aortic dissection in both treatment groups [Suppl. table 1].

The incidence of in-hospital events were: acute coronary syndrome 3.7% (endovascular n=3, medical treatment alone n=2), neurological complications 7.4% (endovascular n=7, medical treatment alone n=3), dialysis 2.9% (endovascular n=3, medical treatment alone n=1), pulmonary infection 11.8% (endovascular n=11, medical treatment alone n=5).

Anatomical features

The anatomical features of the cohort are shown in Table 2 and Suppl. Table 2. At presentation the maximum aortic diameter was greater in the endovascularly treated group compared with the group treated with medical treatment alone. In both groups the dissection tended to involve both the thoracic and abdominal

aorta (endovascular group 93%, medical treatment only group 81.7%). In the endovascularly treated group the true lumen tended to be smaller than the false lumen, whereas in the group that received medical treatment only the true lumen was greater than the false lumen. In both treatment groups a large proportion of the patients had a patent false lumen and there was no difference in the number of entry tears. The size of the largest entry tear was significantly greater in the endovascular treatment group.

At 2-years follow-up there was an average increase in the true lumen diameter of the thoracic aorta of 8mm in the endovascular treatment group with no corresponding increase in the true lumen diameter of the abdominal segment in this group. The majority of the patients in the medically treated group had partial (51.7%) or complete (34.5%) thoracic aortic false lumen thrombosis whereas in the endovascularly treated group approximately one third of patients had patency of the false lumen. At 5-years the average size of the false lumen in the endovascularly treated group had increased compared to the 2-year data and only 27.3% of patients had complete thrombosis of the thoracic aortic false lumen following endovascular treatment.

Survival analysis

The mean follow up in the cohort was 51 (1-132) months; thirty-three deaths, 8 aortic-related, 10 cardiac, 10 cancer-related and 5 other occurred, and 48 aortic events were recorded. The 30-day all cause survival and aortic event-free survival were 98.5% (94.3-99.6%) and 94.8% (89.4-97.5%) respectively. Cumulative survival was 94.0% (95% CI 88.4 - 97.0%) at one year and 74.8% (64.5-82.5%) at five years [Figure 1]. The aortic event-free survival at one year

and at five years was 75.6% (67.3-82.1%) and 58.7% (48.1-67.8%) respectively [Figure 2].

There was no difference in all cause survival at one and five years between the patients treated endovascularly and those receiving medical treatment alone (HR=0.99 (0.49-2.02), p=0.996). There was no difference in aortic event-free survival at one and five years between the patients treated endovascularly and those receiving medical treatment alone (HR=1.33 (0.78-2.34), p=0.329).

Factors associated with overall and aortic event free survival

Patient characteristics, cardiovascular risk factors and imaging features, and their association with all-cause survival, are summarised in supplementary tables 3, 4 and 5 respectively. Age was significantly associated with all cause survival with an increase in hazard of death of 26% for every 5 years increase in age (HR=1.26 (1.08-1.46), p=0.003) [Suppl. Figure 1]. There was no association between cardiovascular parameters, CRP, eGFR and the amount of hypertensive medication on admission and all cause mortality. There was an inverse relationship between the amount of anti-hypertensive medication on discharge and survival. An increase of one drug in the number of antihypertensive medications prescribed resulted in a decrease in hazard of death of 32% (HR=0.68 (0.53-0.88), p=0.013) [Suppl. Figure 2]. Aortic event-free survival data is presented in supplementary tables 6, 7 and 8. Taller patients were more likely to experience an aortic event during follow up; with a one centimetre increase in height associated with an increase in the hazard of experiencing an aortic event of 5% (HR=1.04 (1.01-1.05), p=0.005) [Suppl. Figure 3]. An increase in the diameter of the largest entry tear [Suppl. Figure 4], the diameter of the

255 descending thoracic aorta [Suppl. Figure 5] and the diameter of the descending
256 thoracic aorta false lumen [Suppl. Figure 6] were all significantly associated with
257 an increased hazard of aortic events during follow up. A one mm increase in both
258 the size of the primary tear and the descending thoracic aorta was associated
259 with a 7% increase in the hazard of an aortic event ((HR=1.07 (1.02-1.11),
260 p=0.003) and (HR=1.07 (1.02-1.11), p=0.002), respectively) and a 5% increase
261 for a 1mm increase in the descending thoracic aorta false lumen (HR=1.05 (1.01-
262 1.09), p=0.008).

Discussion

This study evaluates the outcomes of patients with type B aortic dissection treated endovascularly in the presence of complications and with best medical therapy in uncomplicated cases. The data at 1- and 5-years of follow-up demonstrate that there is no difference in all cause survival and aortic event-free survival between these two groups. Survival has traditionally been worse in patients with complicated compared with uncomplicated type B dissection, with survival figures in the region of 56.3-87% and 70.2-89% respectively at 5 years.(7) Better outcomes can be achieved in complicated patients by early identification and active management of complications, a low rate of procedural mortality and morbidity and active management of patients during follow using blood pressure control, surveillance imaging and timely re-intervention when required.(1, 3, 8)

The aim of thoracic endovascular treatment is to cover the proximal entry tear to direct aortic blood flow towards the true lumen, to induce false lumen thrombosis and positive aortic remodelling, with the intended benefit of improving survival. It is thought that early endovascular treatment is likely to result in the maximum amount of aortic remodelling, because the aorta still has plasticity, and therefore result in the best long-term outcomes. Thoracic endovascular repair in the acute setting is associated with a relatively high risk of retrograde type A aortic dissection.(9) In the context of life-threatening conditions such as rupture and visceral malperfusion this risk is considered acceptable. However in the absence of non-life threatening complications or

when considering prophylactic treatment of type B aortic dissection this risk must be carefully evaluated.

The data in this manuscript suggest that thoracic endovascular aortic repair is not able to prevent all aortic events during follow up, which is one of the primary aims of this treatment. Data from the INSTEAD trial also shows at up to 52 months, the mean follow-up in this series, a continued incidence of aortic events (4) and the consensus document also has similar findings.(4, 10, Nienaber, 2013 #8090) Techniques other than thoracic endovascular repair are available and should be considered in the management of patients with chronic type B aortic dissection to try to augment the effect of endovascular repair. These include extension of the aortic endografting into the abdominal segment using branched and fenestrated devices, placement of endovascular coils and plugs (candy-plug) in the false lumen, occlusion of the false lumen by ballooning a stent graft in the true lumen to prevent retrograde flow (knickerbocker technique) and the STABILISE technique.(11-13)

A more in depth evaluation of the anatomy in these patients demonstrates that patients in the group with complications treated endovascularly tended to have a larger primary entry tear, a larger starting aortic diameter, and a narrower true lumen compared with the group treated with medical treatment alone. Patients in the medically treated group tended to have a larger true compared with false lumen. These features suggest that the pressure in the false lumen of patients with complications on presentation may be greater than in the group treated medically. Following TEVAR thoracic but not abdominal aortic remodelling was

seen, which is consistent with the results of other series.(14) Approximately one third of patients in the endovascular group did not have false lumen thrombosis in the thoracic aorta at 2 years, and this led to a progressive increase in thoracic aortic diameter during follow-up. This residual flow in the false lumen in these patients may have influenced the incidence of aortic events during follow-up.(15) The long follow-up in this series allows a particular evaluation of false lumen thrombosis in type B aortic dissection the over time.

Uncomplicated patients with type B aortic dissection typically follow a varied course following presentation. Some centres now advocate high frequency serial imaging (~3 CT angiograms) in the first ten days following presentation to try to identify patients early that are likely to undergo rapid development of aortic complications. In this series we have shown that anatomic features such as a large proximal entry tear, a large descending thoracic aortic diameter and a large diameter of the descending thoracic aortic false lumen on presentation are related to an increased hazard of experiencing an aortic event during follow-up. Taller patients were also more likely to experience an aortic event during follow up and may represent a group with undiagnosed connective tissue disease.(16) The effect of height on outcomes was significant, with a 10cm increase in height resulting in a 48% increase in the hazard of experiencing an aortic event during follow up. Blood pressure control was shown to have a significant effect on all-cause survival, with more active management related to better outcomes.

The International Registry of Aortic Dissection (IRAD) contains data collected from centres across the world and represents a unique resource to study the

diagnosis and management of patients with aortic dissection.(17) One of the limitations of the registry however is the heterogeneity of the data, which reflects the local clinical management of patients and local CT image interpretation in each of the centres. One of the strengths of the series described in the current manuscript is that patients were treated in a single centre, with a standardised procedure for clinical management and image interpretation. During the course of the study there was an increase in the level of clinical and surgical experience and this is one of the limitations of this study, also data on re-intervention and stent graft type was not collected. The image analysis was limited in that inter- and intra-observer reproducibility was not specifically performed and thrombosis was assumed to be present when contrast in the false lumen was absent; standard clinical acquisition protocols were used but these can over-represent the amount of thrombosis if low-flow states are present.

In conclusion, active management of patients with type B aortic dissection results in good long-term survival despite presenting features that have traditionally been associated with adverse outcomes. All patients require close lifetime surveillance as aortic events, even after endografting, continue to occur during follow-up.

360 **References**

- 361 1. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H,
362 et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases:
363 Document covering acute and chronic aortic diseases of the thoracic and
364 abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of
365 Aortic Diseases of the European Society of Cardiology (ESC). *Eur Heart J*.
366 2014;35:2873-926.
- 367 2. Acosta S, Blomstrand D, Gottsater A. Epidemiology and long-term
368 prognostic factors in acute type B aortic dissection. *Ann Vasc Surg*. 2007;21:415-
369 22.
- 370 3. Coady MA, Ikonomidis JS, Cheung AT, Matsumoto AH, Dake MD, Chaikof
371 EL, et al. Surgical management of descending thoracic aortic disease: open and
372 endovascular approaches: a scientific statement from the American Heart
373 Association. *Circulation*. 2010;121:2780-804.
- 374 4. Nienaber CA, Kische S, Rousseau H, Eggebrecht H, Rehders TC, Kundt G, et
375 al. Endovascular repair of type B aortic dissection: long-term results of the
376 randomized investigation of stent grafts in aortic dissection trial. *Circ Cardiovasc*
377 *Interv*. 2013;6:407-16.
- 378 5. van Bogerijen GH, Tolenaar JL, Rampoldi V, Moll FL, van Herwaarden JA,
379 Jonker FH, et al. Predictors of aortic growth in uncomplicated type B aortic
380 dissection. *J Vasc Surg*. 2014;59:1134-43.
- 381 6. Mid-term outcomes and aortic remodelling after thoracic endovascular
382 repair for acute, subacute, and chronic aortic dissection: the VIRTUE Registry.
383 *Eur J Vasc Endovasc Surg*. 2014;48:363-71.
- 384 7. Nienaber CA, Clough RE. Management of acute aortic dissection. *Lancet*.
385 2015;385:800-11.
- 386 8. Riambau V, Bockler D, Brunkwall J, Cao P, Chiesa R, Coppi G, et al. Editor's
387 Choice - Management of Descending Thoracic Aorta Diseases: Clinical Practice
388 Guidelines of the European Society for Vascular Surgery (ESVS). *Eur J Vasc*
389 *Endovasc Surg*. 2017;53:4-52.
- 390 9. Canaud L, Ozdemir BA, Patterson BO, Holt PJ, Loftus IM, Thompson MM.
391 Retrograde aortic dissection after thoracic endovascular aortic repair. *Ann Surg*.
392 2014;260:389-95.
- 393 10. Fattori R, Cao P, De Rango P, Czerny M, Evangelista A, Nienaber C, et al.
394 Interdisciplinary expert consensus document on management of type B aortic
395 dissection. *J Am Coll Cardiol*. 2013;61:1661-78.
- 396 11. Kolbel T, Carpenter SW, Lohrenz C, Tsilimparis N, Larena-Avellaneda A,
397 Debus ES. Addressing persistent false lumen flow in chronic aortic dissection: the
398 knickerbocker technique. *J Endovasc Ther*. 2014;21:117-22.
- 399 12. Kolbel T, Lohrenz C, Kieback A, Diener H, Debus ES, Larena-Avellaneda A.
400 Distal false lumen occlusion in aortic dissection with a homemade extra-large
401 vascular plug: the candy-plug technique. *J Endovasc Ther*. 2013;20:484-9.
- 402 13. Hofferberth SC, Nixon IK, Boston RC, McLachlan CS, Mossop PJ. Stent-
403 assisted balloon-induced intimal disruption and relamination in aortic dissection
404 repair: the STABILISE concept. *J Thorac Cardiovasc Surg*. 2014;147:1240-5.
- 405 14. Sayer D, Bratby M, Brooks M, Loftus I, Morgan R, Thompson M. Aortic
406 morphology following endovascular repair of acute and chronic type B aortic

407 dissection: implications for management. Eur J Vasc Endovasc Surg.
408 2008;36:522-9.

409 15. Tsai TT, Evangelista A, Nienaber CA, Myrmel T, Meinhardt G, Cooper JV, et
410 al. Partial thrombosis of the false lumen in patients with acute type B aortic
411 dissection. N Engl J Med. 2007;357:349-59.

412 16. Weinsaft JW, Devereux RB, Preiss LR, Feher A, Roman MJ, Basson CT, et al.
413 Aortic Dissection in Patients With Genetically Mediated Aneurysms: Incidence
414 and Predictors in the GenTAC Registry. J Am Coll Cardiol. 2016;67:2744-54.

415 17. Pape LA, Awais M, Woznicki EM, Suzuki T, Trimarchi S, Evangelista A, et
416 al. Presentation, Diagnosis, and Outcomes of Acute Aortic Dissection: 17-Year
417 Trends From the International Registry of Acute Aortic Dissection. J Am Coll
418 Cardiol. 2015;66:350-8.

419

420

421

422 **Tables**

	All	Endovascular treatment	Medical treatment only	p- value
Total	136	64	72	
Age, mean(sd)	61.7(13.2)	58.5(12.7)	64.5(13.1)	0.008
Male sex, %	77.2	79.7	75.0	0.515
Height (cm), mean(sd)	173.5(9.7)	174.6(8.9)	172.6(10.3)	0.264
Hypertension, %	71.3	75.0	68.1	0.371
Diabetes, %	9.6	9.9	9.4	0.924
Dyslipidaemia, %	25.0	17.2	31.9	0.047
BMI, median(IQR)	27(24.2- 31.0)	27.0(24.0- 31.4)	26.9(24.4- 29.7)	0.945
Coronary artery disease, %	5.9	4.7	7.0	0.721
Co-existing AAA, %	9.6	10.9	8.5	0.625
Previously treated AAA, %	4.4	0	8.5	0.029
Renal insufficiency, %	0	0	0	
Connective tissue disease, %	5.9	9.4	2.8	0.149
Family history of Marfan, %	3.7	4.7	2.8	0.668

423

424

425

426

Table 1.
Patient characteristics

	All	Endovascular treatment	Medical treatment only	p-value
<u>Presentation</u>				
Maximum aortic diameter, median(IQR)	38(35-43)	41(36-45)	37(34-40)	0.003
Dissection involving thoracic and abdominal aorta, %	87.2	93.0	81.7	0.096
Total diameter of the thoracic aorta, median(IQR)	37(34-42)	39(34-42)	36(32.5-38)	0.006
True lumen diameter in the thoracic aorta, median(IQR)	18(14-23)	18(15-24)	19.5(12.5-23)	0.900
False lumen diameter in the thoracic aorta, median(IQR)	18(12-26)	20(14-28)	17(10-23)	0.060
False lumen status, %				
Patent	52.6	59.7	45.8	0.105
Partially thrombosed	38.8	36.8	40.7	
Completely thrombosed	8.6	3.5	13.6	
<u>2 years</u>				
Maximum aortic diameter, median(IQR)	40.5(36-44.5)	42(38-47)	39(35-42)	0.088
Total diameter of the thoracic aorta, median(IQR)	40(36-43)	41(36-44)	38(35-41)	0.067
True lumen diameter in the thoracic aorta, median(IQR)	25(15-30)	25.5(16-30)	21(13-28)	0.180
False lumen diameter in the thoracic aorta, median(IQR)	18(8-27)	18.5(8-28)	18(8-26)	0.665
False lumen status, %				
Patent	24.3	31.7	13.8	0.184
Partially thrombosed	41.4	34.2	51.7	
Completely thrombosed	34.3	34.2	34.5	
<u>5 years</u>				
Maximum aortic diameter, median(IQR)	40(37-47)	42(38-48)	37.5(33-41.5)	0.045
Total diameter of the thoracic aorta, median(IQR)	40.5(36-44.5)	42(38-47)	39(35-42)	0.088
True lumen diameter in the thoracic aorta, median(IQR)	23(16-31)	20(16-32)	28(18-30)	0.600
False lumen diameter in the thoracic aorta, median(IQR)	21(8.5-28)	23(12-28)	14(4-34)	0.316
False lumen status, %				
Patent	28.6	39.4	6.3	0.049
Partially thrombosed	38.8	33.3	50.0	
Completely thrombosed	32.7	27.3	43.8	

Table 2.
Anatomical features of the patient cohort

Figure legends

Figure 1: Kaplan Meier estimate demonstrating 94.4% and 75.5% cumulative overall survival at one and five years respectively

Figure 2: The aortic event-free survival at one year and at five years was 75.5% and 58.0% respectively